ACCESSORY ANTEROLATERAL TALAR FACET AS AN ETIOLOGY OF PAINFUL TALOCALCANEAL IMPINGEMENT IN THE RIGID FLATFOOT: A NEW DIAGNOSIS

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ABSTRACT

A retrospective review identified six patients with seven painful rigid flatfeet. In each case, pain was localized laterally to an accessory facet of the anterolateral talus. Cross-sectional imaging demonstrated no evidence of tarsal coalition. In five of the six, preoperative magnetic resonance imaging (MRI) was obtained and in each case demonstrated focal abutting bone marrow edema consistent with impingement between the accessory facet and the anterior calcaneus.

Seven feet in six patients underwent resection of the accessory facet with additional subtalar joint-sparing reconstructive procedures. At an average follow-up of 11 months, clinical results were graded as four good and two fair.

An association between this accessory facet and pain in the rigid flatfoot has not been previously reported. Obesity was universal and may represent a risk factor for facet impingement. At early follow-up, facet resection with subtalar joint-sparing flatfoot reconstruction provided good results with symptomatic and functional improvement in the majority of patients.

INTRODUCTION

Primary flatfoot deformity is a common source of referral in orthopaedic surgery. The distinction between the flexible and rigid flatfoot is important for diagnosis and treatment. The painful, rigid flatfoot requires further investigation. A rigid flatfoot is distinguished from the flexible variety by physical exam. Subtalar motion is restricted and attempted motion may be painful. Peroneal spasm or contracture may be evident with attempted passive inversion. The toe-rise test is performed with difficulty and the longitudinal arch does not reform. Medial midfoot callosities may be present due to prominence of the talar head or navicular.

Radiographs and computed tomography (CT) with coronal and sagittal reconstructions are useful to identify tarsal coalitions, the most common etiology of the rigid flatfoot in adolescents and young adults. Computed tomography or magnetic resonance imaging may assist in detecting incomplete coalitions (cartilaginous or fibrous). Other established etiologies of the rigid flatfoot include infectious, inflammatory, or degenerative arthritides, neoplastic or neurologic processes, and osteochondral fractures. Laboratory studies or radioscintigraphy may help in making diagnoses such as osteoid osteoma or inflammatory arthritides.

Most patients with a symptomatic, rigid flatfoot will have an identifiable causation. However, there are several reports of idiopathic rigid flatfoot in the literature.[1-3] These authors have reported that the idiopathic rigid flatfoot is difficult to treat and is often recalcitrant to conservative and operative measures.[2]

We present a retrospective review of patients with rigid flatfoot deformity treated operatively for painful talocalcaneal impingement associated with an accessory facet of the anterolateral talus. The clinical presentation, radiographic studies, and treatment course in this population are reviewed. Our hypothesis was that accessory facet resection combined with subtalar joint-sparing reconstructive procedures for residual deformity would provide improvement in symptoms.

MATERIALS AND METHODS

All patients treated operatively by a single surgeon (JEF) from 2000 to 2005 for primary rigid flatfoot in association with an accessory anterolateral talar facet were retrospectively reviewed. This study was conducted with Institutional Review Board approval. Hospital charts and imaging studies were reviewed for each patient. Inclusion criteria included pediatric patients with a history of hindfoot pain and primary flatfoot deformity; physical examination demonstrating painful rigid flatfoot deformity, lateral hindfoot pain upon attempted passive hindfoot eversion, and tenderness in the sinus tarsi; and radiographic studies demonstrating the absence of tarsal coalition and the presence of an accessory anterolateral talar facet. Exclusion criteria included age greater than 18 years at presentation (two patients), nonoperative

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TABLE 1. Comfort and Johnson Outcome Score[4]

Points	Pain	Function	Subtalar Motion	Outome Score (Rating)
3	None	Unlimited activity	-	7 (Excellent)
2	Slight (with sports/running)	Limited sports/running	-	5-6 points (Good)
1	Moderate (walking, ADL,* light activity)	Walking, ADL,* light activity	Present	3-4 points (Fair)
0	Severe (no relief, same as preoperative)	No improvement over preoperative	None	0-2 points (Poor)

(*ADL - activites of daily living)

management (three patients), an accessory facet associated with tarsal coalition (four patients), or an accessory facet associated with subtalar arthrosis (two patients). Data collected included age, symptom duration, radiographic findings, treatment method, and response at most recent follow-up. Outcomes were graded by the seven-point postoperative outcome score described by Comfort and Johnson in their report of the results of talocalcaneal coalition resection.[4] This outcome measure grades postoperative pain, function, and subtalar motion describing the clinical result as excellent, good, fair, or poor (Table 1).

The diagnostic evaluation included physical examination, standing radiographs, and cross-sectional imaging (CT and/or MR). Most patients were referred after having been treated with multiple non-operative interventions by another physician. Initial management included physical therapy, a trial of immobilization, orthotics, and non-steroidal anti-inflammatory medications. Surgical intervention was offered when pain persisted despite non-operative treatment. Maximal pain was localized to the sinus tarsi in all patients. The principle of preserving the subtalar joint and avoiding subtalar arthrodesis guided operative treatment. Talocalcaneal impingement was treated with resection of the accessory anterolateral talar facet; residual deformity, in these rigid flat feet, was addressed with subtalar joint-sparing flatfoot reconstruction. Deformity correction was approached stepwise, e.g., adding additional procedures as required for residual deformity: Gastrocnemius recession first; peroneal lengthening second. After gastrocnemius recession and peroneal lengthening, all subtalar joints became mobile intra-operatively. If necessary, medial displacement calcaneal osteotomy for hindfoot valgus and/or lateral column lengthening for forefoot abduction were then performed.

OPERATIVE TECHNIQUE

The operative technique of resecting the accessory anterolateral talar facet was performed through a longitudinal incision centered over the sinus tarsi. This incision was made from the posterior tip of the fibula and directed distally toward the base of the fourth metatarsal. The incision was extended proximally along the

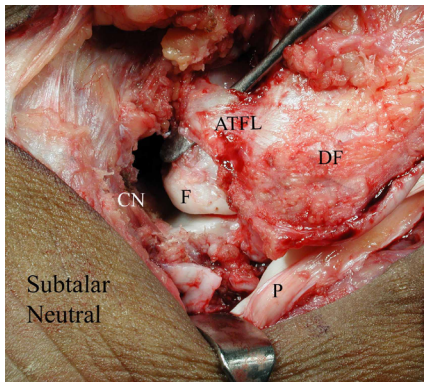


Figure 1a. Accessory anterolateral talar facet (F) in neutral hindfoot alignment.

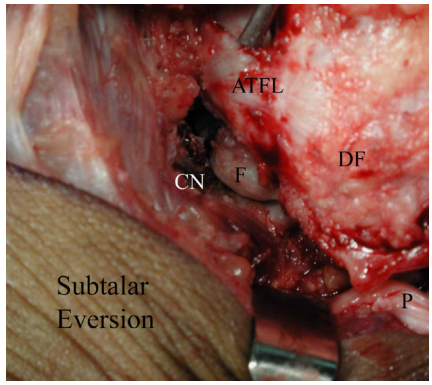


Figure 1b. Impingement of the accessory facet (F) on the calcaneal neck (CN) with hindfoot eversion.

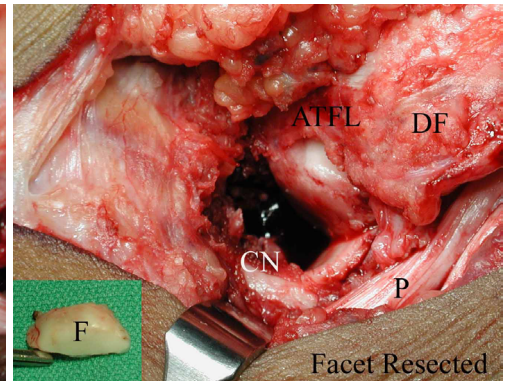
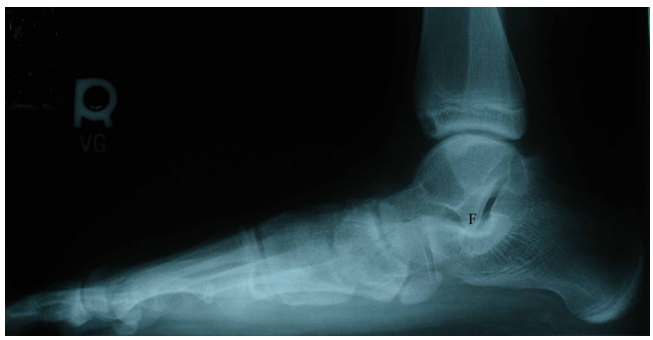


Figure 1c. Decompression of the sinus tarsi following accessory facet resection

Figure 1. Intraoperative photos of a 16-year-old patient. (F - accessory anterolateral talar facet, ATFL – anterior talofibular ligament, P – peroneal tendons, DF – distal fibula, CN – calcaneal neck)



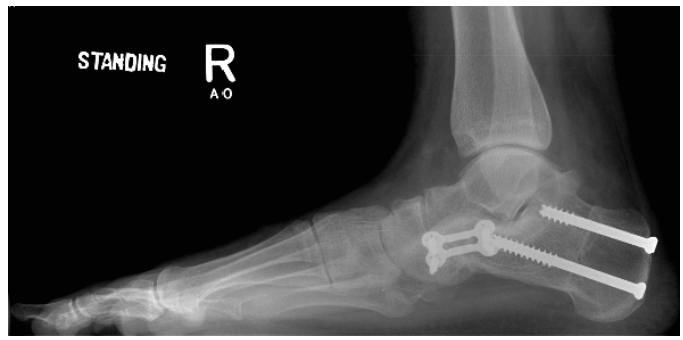


Figure 2. Clinical example. Figure 2a. (left) Preoperative radiograph (F – accessory facet). Figure 2b. (right) Postoperative radiograph following accessory facet resection, medial displacement calcaneal osteotomy, and calcaneocuboid distraction arthrodesis.

posterior border of the fibula when performing peroneal lengthening. The incision was deepened dorsal to the peroneal tendons below the fibula and in each case a bony protrusion with a cartilaginous cap was found filling the sinus tarsi. This originated from the lateral talar process just below the inferior aspect of the anterior talofibular ligament (ATFL) and extended distally, abutting the calcaneus. This accessory anterolateral talar facet was resected in a semi-coronal plane with an osteotome, perpendicular to the posterior facet articular surface and flush with the insertion of the anterior talofibular ligament on the lateral process of the talus. The resulting cancellous surface was rasped smooth and impregnated with bone wax (Figure 1). In patients with contractures, a gastrocnemius recession and/or peroneal Z-lengthening above the superior peroneal retinaculum was performed. Remaining planovalgus deformity was corrected with a medial displacement calcaneus osteotomy and/or lateral column lengthening with calcaneocuboid distraction arthrodesis (Figure 2).

The series consisted of six patients (seven feet) with painful rigid flatfoot, an accessory facet, and no tarsal coalition by CT and/or MR imaging (idiopathic rigid flatfoot). Accessory facets were present bilaterally in all four of the patients with bilateral imaging studies; the facet was symptomatic bilaterally in two of these four patients. The average age at presentation was 15.0 years (range 13-17 years) with average symptom duration of 1.8 years prior to presentation. All six patients were male. The average body mass index (BMI) was 34.6 (range 29.8–44.5) (Table 2).

RESULTS

All patients were evaluated with radiographs and cross-sectional imaging (CT and/or MR). The lateral radiograph often suggested an accessory anterolateral talar facet with broadening of the anteroinferior lateral talar process; sometimes the facet was difficult to appreciate on plain radiographs due to the semi-coronal plane of the lateral talar process and associated hindfoot deformity (Figure 3a). Associated dorsal talar beaking was observed in four of the six patients. CT multiplanar reformats assisted in defining the accessory facet (Figure 3b). In five of the six patients, MR imaging was obtained and demonstrated abutting bone marrow edema between the talus and calcaneus, on sagittal fat-saturated T2-weighted images, in all five cases (Figure 4). This was localized to the accessory anterolateral talar facet and the adjacent calcaneus anterior to the posterior facet. This edema was interpreted as consistent with talocalcaneal impingement. Intraoperatively, the accessory facet was noted to have a hyaline cartilage surface, often with mild degenerative changes. Synovitis was also present within the sinus tarsi. Histologic evaluation demonstrated

TABLE 2. Demographics

Case	Age at Presentation (years)	Symptom Duration (years)	Body Mass Index	Gender	Symtomatic Accessory Facet	Bilateral Accessory
1	13.3	1.0	29.9	M	Right	Yes
2	14.3	0.8	44.5	M	Bilatral	Yes
3	16.7	3.6	40.9	M	Left	Unknown
4	14.5	0.7	30.5	M	Bilateral	Yes
5	17.4	3.6	29.8	M	Left	Unknown
6	13.9	1.2	31.9	M	Right	Yes

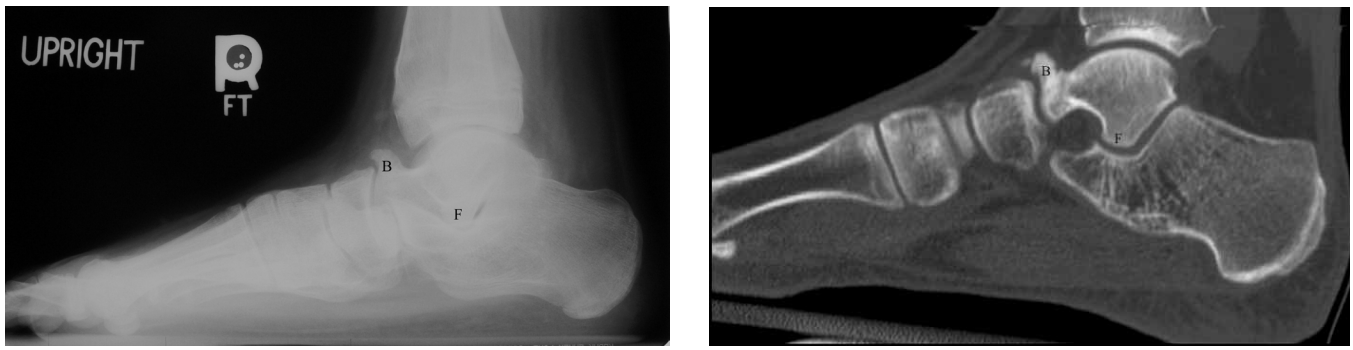


Figure 3. Clinical example. Figure 3a. (left) Lateral radiograph of accessory anterolateral talar facet (F – accessory facet, B – dorsal talar beak). Figure 3b. (right) CT scan (F – accessory facet, B – dorsal talar beak).

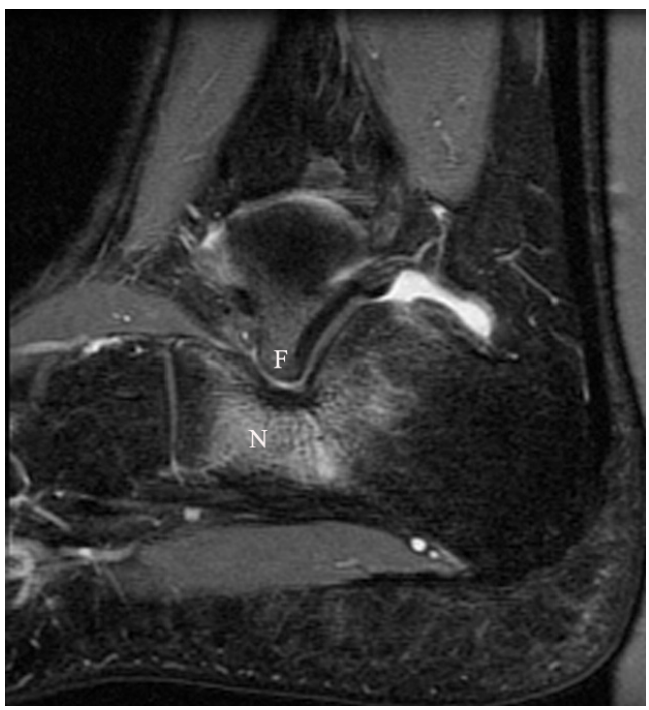


Figure 4. MR imaging demonstrating abutting bone marrow edema between the accessory facet (F)) and the calcaneal neck (N).

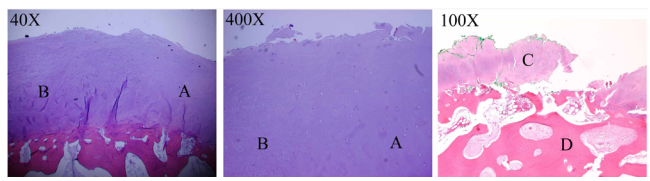


Figure 5. Histologic evaluation of excised accessory facet with hematoxylin and eosin (A – normal hyaline cartilage, B – early fibrocartilaginous change, C – thin, fissured cartilage, D – thickened subchondral bone with cysts).

that within the resected facet, there were regions of normal hyaline cartilage intermixed with areas of thin, fissured cartilage, early fibrocartilaginous changes, and thickened subchondral bone with microscopic cyst formation (Figure 5). Continuity between the articular surface of the posterior facet of the talus and the accessory facet was noted both on MR and clinically at the time of surgery. No tarsal coalitions (osseous or fibrous) were observed.

All six patients underwent resection of the accessory facet; one bilaterally. One was treated with isolated facet resection; one with unilateral facet resection and peroneal lengthening; one with bilateral facet resection, gastrocnemius recession, and peroneal lengthening; and three with unilateral facet resection, gastrocnemius recession, peroneal lengthening, calcaneal osteotomy, and lateral column lengthening (Table 3).

TABLE 3. Treatment Methods and Outcomes

Case	Symptomatic Accessory Facet	Accessory Facet Resection	Additional Procedures 1-gastrocnemius recession 2-peroneal lengthening 3-calcaneal osteotomy 4-lateral column lengthening	Postoperative Follow-up	Outcome Score (Rating)
1	Right	Right	1,2,3,4	12 months	3 (Fair)
2	Bilateral	Bilateral	1,2	28 months	4 (Fair)
3	Left	Left	None	8 months	5 (Good)
4	Bilateral	Left	1,2,3,4	4 months	5 (Good)
5	Left	Left	1,2,3,4	5 months	5 (Good)
6	Right	Right	2	9 months	6 (Good)

Outcomes were graded with the seven-point scoring system as described by Comfort and Johnson.[4] There were no excellent results (outcome score of seven). Four patients had a good result (score of five to six) and two had a fair result (with a score of three to four). The fair results occurred in the two patients with the longest clinical follow-up (12 and 28 months). There were no poor results. The average pain and function scores were 2.2 and 1.7, respectively. Five of the six patients had subtalar motion postoperatively that was not present preoperatively. There were no complications in this series. The average postoperative follow-up was 11 months (range 4 months to 28 months); one patient was lost to follow-up (Table 3).

DISCUSSION

The association between the accessory anterolateral talar facet and symptomatic rigid flatfoot has not been previously reported. This anatomic variant represents a new etiology for painful talocalcaneal impingement in the rigid flatfoot. Cross-sectional imaging is useful for identifying the anomaly, and MRI can demonstrate evidence of impingement with localized bone marrow edema. In our patients with an isolated accessory facet, facet resection with subtalar-sparing flatfoot reconstruction provided good results with early pain relief and a return to function in four of six patients.

The stiff, painful flatfoot was first described as the "peroneal spastic" flatfoot by Sir Robert Jones in 1897.[1] The "spastic" flatfoot does not require a constant state of peroneal contraction, although active spasm may be produced by irritation of the painful hindfoot with motion and weight bearing. Chronic subtalar eversion may result in adaptive shortening of the peroneal musculature. The term "rigid flatfoot" is now preferred for description of the foot with limited subtalar motion and planus deformity.[2,5,6] The link between structural anomalies of the tarsus and rigid flatfoot was established by Slomann in 1921, Badgley in 1927, and Harris and Beath in 1948.[7-9] In a study of 3600 Canadian Army recruits, Harris and Beath found a 2% incidence of spastic (rigid) flatfoot and a 6% incidence of flexible flatfoot.[8] Tarsal coalitions were the underlying etiology in 88% of the rigid deformities; two-thirds of the coalitions were talocalcaneal and one-fifth were calcaneonavicular.[8]

Radiographic features classically associated with tarsal coalition include dorsal beaking of the talar neck, narrowing of the posterior talocalcaneal joint, and broadening of the lateral talar process.[10] These findings have been observed in the rigid flatfoot without tarsal coalition.[2] In the present study, four of six patients demonstrated dorsal talar beaking on the lateral radiograph. Dorsal beaking of the talar neck is theorized to result from restricted subtalar motion leading to excessive motion of the talonavicular joint and subsequent gradual ossification of the elevated talar neck periosteum.[11] Molding of the talar head by the navicular as a result of the increased talonavicular motion has also been suggested as a mechanism.[12] Cowell stated that the broadening of the lateral talar process in the patient with tarsal coalition was a secondary finding resulting from impingement of the lateral process of the talus upon the sulcus calcaneus as the calcaneus is forced into valgus.[6,11]

Patients with painful rigid flatfoot in the absence of tarsal coalition or systemic abnormality have been described by multiple authors.[1,2,5,8,13] These reports suggest that the idiopathic rigid flatfoot is difficult to manage and treatment options are limited when standard conservative measures fail to provide relief. In 1961, Braddock reported on 28 patients with 43 symptomatic peroneal spastic flatfeet, evaluated with anteroposterior, lateral, oblique, and Harris-Beath radiographs.[14] In this series there were 27 feet with "normal" radiographs (anteroposterior, oblique, lateral and axial; however no cross-sectional imaging), although "slight beaking" of the talus was included in the normal group. Patients were treated with manipulation under anesthesia, a walking cast, and/or an iron with T-strap. Interestingly, the "normal"-radiograph group (without coalition) had greater initial pain and disability than patients with positive radiographs (coalition), and 4 of the 27 normal feet had long-term subtalar rigidity. In 1974, Rankin presented a series of 24 military recruits with rigid flatfoot evaluated with plain radiographs only.[15] Seven of the 24 patients were without tarsal coalition. In all patients, symptoms subsided upon withdrawal from basic training; although the patients were followed for an average of only one month after the diagnosis of rigid flatfoot.

Luhmann reported a series of 13 idiopathic rigid flatfeet in nine patients.[2] Two of the 13 feet had dorsal talar beaking, and tarsal coalition was not identified by CT or MR in any of the 13 feet. Our review of the radiographs and CT images from Luhmann's publication suggests that some of these patients had accessory facets. All patients underwent examination under anesthesia with subtalar injection (methylprednisolone and/or bupivacaine). Improved motion was noted intraoperatively in nine feet; the remaining four feet were treated with fractional peroneal lengthenings. All patients were casted in maximal inversion for three weeks. Outcomes at an average 18-month follow-up demonstrated five feet with sustained pain relief and eight feet with persistent pain. There was no correlation of the clinical results with the treatment method. Luhmann theorized that the rigid flatfoot in their series was a progression from the flexible flatfoot of childhood to a rigid planovalgus deformity

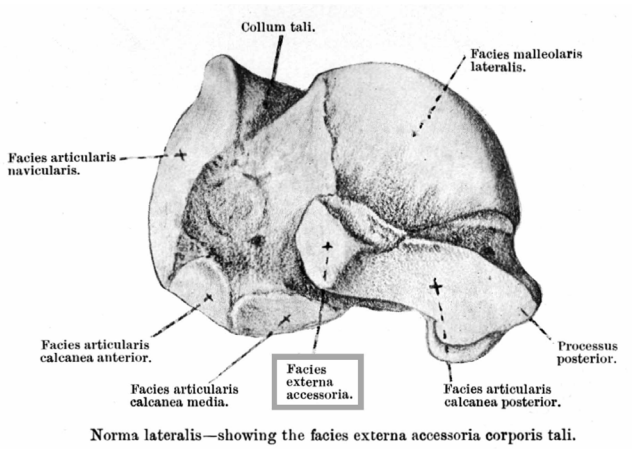


Figure 6. Original description of the accessory anterolateral talar facet ***(facies externa accessoria)*****.**[21]

secondary to obesity. All of the patients in the series were greater than the 75th percentile of weight for age, and seven of the nine patients had weights greater than the 95th percentile. The six patients in our series with idiopathic rigid flatfoot who required accessory facet resection had an average body mass index (BMI) of 34.6 (normal range is 18.5-25). Four patients had a BMI less than 32, but two of the six had a BMI greater than 40 which is defined as "extreme obesity".[16] Luhmann also implicated "malicious malalignment" (combined femoral retroversion and external tibial torsion) as an etiologic factor where increased foot progression angle leads to a "medial rollover" gait with minimal passive ankle dorsiflexion and eventual equinovalgus contracture.

The anatomy of the talocalcaneal articulation has been studied and significant anatomic variation exists.[17-20] The accessory facet of the anterolateral talus was first described by Sewell, in a 1904 study of 1006 Egyptian tali. He characterized a number of variants of talar anatomy, including the accessory anterolateral talar facet which he termed *facies externa accessoria corporis talus.* He found this to be present in 10.2% of the tali examined.[21] Figure 6 is a diagram from Sewell's original paper demonstrating the accessory facet originating from the anterior aspect of the lateral talar process inferior to the anterior talofibular ligament. The diagram also suggests continuity of the accessory facet with the posterior facet articular surface, which is a feature we noted in all of our cases. Interestingly, the specimen depicted in this diagram also has a dorsal talar beak (Figure 6). Through an osteologic study of 100 tali, Sarrafian identified large accessory anterolateral talar facets in 4% and small, variable-sized facets in 34%.[22]

The biomechanics of the subtalar joint may explain the development of symptomatic impingement between the accessory anterolateral talar facet and the anterior process of the calcaneus. At foot strike, the subtalar joint is partially everted, which is maximized by the point of flatfoot.[23] In this portion of gait, the subtalar joint externally rotates approximately six degrees in the normal foot and 12 degrees in the flatfoot due to the greater horizontal inclination of the subtalar joint in the flatfoot.[24] Mosca described that the flatfoot deformity has excessive subtalar eversion with altered relationships of the calcaneus, talus, and navicular.[25] The increased subtalar eversion characteristic of flatfoot deformity permits impingement of the accessory facet on the anterior process of the calcaneus and subsequent facet degeneration. A study of CT scans in a group of adult patients with acquired symptomatic flat feet found evidence of talocalcaneal impingement in the sinus tarsi in 92% of 76 scans, and none in a control group of 20 normal feet. The bony changes noted by the authors included direct bone abutment between the talus and calcaneus with sclerosis and cystic changes in the sinus tarsi. They did not note any cases with an abnormal prominence of the anterolateral talus or a cartilaginous cap.[26] This suggests that there are two etiologies of talocalcaneal impingement: a primary impingement caused by the presence of an accessory facet in a pediatric or young adult rigid flatfoot deformity, and a secondary impingement due to acquired subtalar joint subluxation seen with advanced acquired adult flatfoot deformity. It seems likely that the onset of symptoms in the patients in the present series is related to increasing body mass with growth, flatfoot deformity, and subsequent accessory facet impingement due to increasing subtalar joint eversion.

In a study reviewing the detection of tarsal coalitions with CT and MRI, Newman described the association between coalitions and bone marrow edema at the margins of the abnormal joints.[27] A similar finding was present in these patients with accessory anterolateral talar facets, with focal abutting bone marrow edema at the articulation between the accessory facet and the anterior process of the calcaneus. This sign was consistently present on MR imaging and correlated with intraoperative demonstration of accessory facet impingement with associated local synovitis. The histologic evaluation of an excised accessory facet from a patient in this series was consistent with impingement and consequent early degeneration of the hyaline cartilage of the facet (Figure 5).

Four patients with an accessory anterolateral talar facet and tarsal coalitions (three talocalcaneal and one calcaneonavicular) were excluded from the present series. These four patients presented with pain localized to the region of the accessory anterolateral talar facet, not the coalition. Direct palpation of the sinus

tarsi could not be considered an indication of subtalar joint pain as the accessory facet prevents access to the joint. This group is interesting to consider, as the presence of the accessory facet may account for failure to achieve pain relief following tarsal coalition resection in some patients. In fact, two of these patients presented with lateral hindfoot pain three years after technically well-erformed tarsal coalition resections. One question that requires further study is whether patients with an accessory anterolateral facet and a tarsal coalition would benefit from resection of the accessory facet at the time of coalition resection. Another critical clinical point is differentiating the location of pain, which helps to identify the painful structure. In the authors' experience, medial pain is typical of fibrous subtalar coalitions, while lateral pain is localized to the sinus tarsi with talocalcaneal impingement or is retrofibular due to peroneal spasm. The presence of lateral hindfoot pain with a medial subtalar coalition may be an indication of lateral impingement, and the possibility of an accessory facet.

The authors have presented a retrospective series of patients with rigid flatfeet in which an accessory anterolateral talar facet was identified, and corresponded to the location of maximal pain (sinus tarsi) as indicated by patient history and confirmed by physical examination. Radiographs and CT imaging delineated the accessory facet and MR imaging demonstrated abutting edema adjacent to the articulation between the accessory facet and the anterior process of the calcaneus. As suggested by Luhmann, there may be an association between obesity and the painful idiopathic rigid flatfoot.[2] Our series indicates that obesity may be associated with rigid flatfoot deformity with symptomatic talocalcaneal impingement from the accessory anterolateral talar facet.

Our experience with the accessory anterolateral talar facet has led to the development of a stepwise subtalar-sparing operative strategy. Although this is a small series, we have been encouraged by our early clinical results with resection of the accessory facet in the absence of radiographic subtalar arthrosis or an associated coalition. Patients with a persistently painful rigid flatfoot have been treated with subtalar or triple arthrodesis in the past. Saltzman has demonstrated universal radiographic tibiotalar degeneration in patients treated with a triple arthrodesis at long-term follow-up.[28] We believe that the loss of subtalar or hindfoot motion at an early age will eventually lead to symptomatic degenerative arthritis of the ankle in adulthood in many patients. In our patients with flatfoot deformity, resection of the accessory facet and subtalar joint-sparing flatfoot reconstruction improved symptoms and allowed for realignment of the hindfoot.

Further study of the association between rigid flatfoot and the accessory anterolateral talar facet is needed, particularly by longer-term follow-up of our patients. Prospective studies to define the natural history of this anatomic variant and the associated pathologies, such as peroneal spasm or contracture, will improve our understanding of this clinical problem.

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